CHRONIC SUBDURAL HAEMATOMA (A Review of 66 Cases)

by

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IF THE presence of a chronic subdural haematoma is suspected, the neurosurgical management of the case can be simple, and may consist of no more than an exploratory burr-hole, which at the same time confirms the diagnosis and cures the condition. Often, however, alternative diagnoses on clinical grounds are likely, and special methods of investigation are preferable. The majority of the cases are admitted to general medical wards, sometimes with rather vague history and complaints, and it falls to the physician to suspect the condition and select suitable cases for special opinion and investigation. This is often not an easy selection to make, and it is in the hope that this selection might be facilitated that we record our more recent experiences in the diagnosis and treatment of patients with chronic subdural haematoma.

MATERIAL AND METHOD

The case records of all adult patients with chronic subdural haematoma treated in the Department of Neurological Surgery, Royal Victoria Hospital, Belfast, from January 1962 to June 1969 were reviewed. The haematoma was regarded as chronic if there was an interval of over three weeks from injury, or when there was no history of head injury (Ecklin et al, 1956; McKissock et al, 1960; Rosenbluth et al, 1962; Gilmartin, 1964).

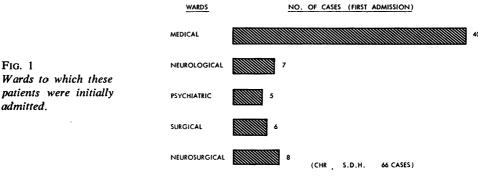
CLINICAL FEATURES

There were 66 patients in this series. It is interesting to note that 52 cases (79 per cent) were first admitted to a medical ward (Fig. 1). Only eight patients were admitted directly to the neurosurgical ward. The age and sex distribution of these patients is given in Table I. There were three times as many males as females.

Table I Age and sex distribution of 66 patients								
Age in Years	11-20	21-30	31-40	41-50	51-60	61-70	71-80	Total
Males	1	3	2	6	8	22	7	49
Females	0	0	1	1	4	8	3	17
Total	1	3	3	7	12	30	10	66

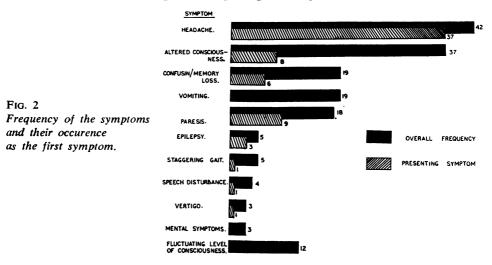
Aetiology

Forty-two patients (61 per cent) had a history of head injury from three weeks to six years previously, though this history was not always available at the time of admission. Two patients were known hypertensives. A history of chronic alcoholism was present in 12 cases. Two patients had idiopathic thrombocytopaenic purpura and one had factor X deficiency. Two cases were known epileptics.



Symptoms

The overall frequency of symptoms and their occurrence as the first symptom are shown in Fig. 2. Over half the cases (56 per cent) presented with headache. It was unilateral in seven cases and was lateralised towards the side of the lesion. One of the characteristic features observed in 12 cases was fluctuation in the level of consciousness. This was helpful in suspecting the diagnosis.



Signs

The frequency of various clinical signs is shown in Fig. 3. Of the 43 cases with altered consciousness, 24 were drowsy, 11 were semi-conscious and eight were comatose. Motor weakness was seen in 40 patients, 24 had hemiparesis, 12 monoparesis and four quadriparesis.

Fig. 3
Frequency of clinical signs.

SIGN	PREQUENCY
ALTERED CONSCIOUSNESS	45
PARESIS	40
BABINSKI'S RESPONSE	27
PUPILLARY	13
DISORIENTATION	10
BRADYCARDIA	9
PAPILLOEDEMA	7
NECK STIFFNESS	5
HEMIANOPIA	5
SPEECH DISTURBANCE	4
CORTICAL SENSORY LOSS	4
DYSDIADOCHOKINESIS	□ 4
LATERAL RECTUS PALSY	4
BILATERAL PTOSIS	3
NYSTAGMUS	<u>[]</u> 1
PURPURIC SPOTS	[] 1

FREQUENCY

Lateralising Value of Signs

- (a) Paresis: Hemiplegia or monoplegia was contralateral to the side of the lesion in 26 cases (73 per cent) and ipsilateral in eight cases (22 per cent); two patients had bilateral haematomata. Of the four patients who had quadriparesis, one had bilateral haematomata, while the other three had a severe degree of brain stem compression.
- (b) *Pupil*: The larger pupil was on the side of the lesion in ten cases (77 per cent), on the contralateral side in two (15 per cent), and on the side of the more bulky haematoma in the one case which had haematomata on both sides.
- (c) Speech Disturbance: All four patients with aphasia or dysphasia had the lesion on the dominant side.
- (d) *Hemianopia*: Visual field defects had accurate localising value in all five cases in which they could be elicited.

INVESTIGATIONS

Plain X-rays of the skull were done in 49 patients. These revealed a fracture of the skull in three cases (6 per cent), and a displaced pineal in 16 cases (32 per cent). The diagnostic value of other ancillary investigations is shown in Table II.

Lumbar Puncture

Cerebrospinal fluid examination was done in eight patients prior to their transfer to the neurosurgical unit. The protein content was slightly elevated in four cases.

However, the procedure is not without its risks, and five patients had definite deterioration in their neurological status following lumbar puncture, necessitating urgent transfer to the neurosurgical unit.

Table II						
Diagnostic accuracy of sp	ecial investigations					

Investigation	Total Number	Correct Localisation	Percentage Correct	
Electroencephalogram	34	18	53	
Angiogram	24	24	100	
Echoencephalogram	24	2 0	83	
Brain Scan (Tc ^{99m})	2	2		
Air Encephalogram	1	1		

TREATMENT AND RESULTS

Treatment

The initial operative procedure in each case was bilateral burr-holes and evacuation of the haematoma. The cavity was reaspirated, if required, more than once. A drain was left in the subdural space in 15 cases. There were eight cases (12 per cent) with bilateral subdural haematomata. A haematoma capsule was present in 34 cases (51 per cent), and in six of these it was described as "thick" or "leathery". Three cases required delayed craniotomy and excision of the membranes when the haematoma failed to resolve after repeated aspirations. Failure of the compressed hemisphere to expand after evacuation was encountered on 20 occasions. In four it was dealt with by intraventricular injection of saline; the remaining 16 had saline injected by the lumbar route. The latter was undoubtedly a more satisfactory procedure. The intracranial hypotension persisted post-operatively in six patients, and lumbar saline injections were repeated in these cases. The expansion of the hemisphere was studied by the simple procedure of applying clips on the surface of the brain and the dura, and following their approximation with serial radiographs.

TABLE III

Results – As a function of the initial level of consciousness

Level of Consciousness	Total No.	Fully Recovered No. %	Slightly Disabled No. %	Severely Disabled No. %	Died No. %
Alert	23	23 100	0 —	0 —	0 —
Drowsy	24	23 96	1 4	0 —	0 -
Semicomatose	11	8 73	0 —	1 9	2 18
Comatose	8	2 25	1 12.5	1 12.5	4 50

TABLE IV

Results – As a function of the age of the patient

Age	Total No.	Fully Recovered No. %	Slightly Disabled No. %	Severely Disabled No. %	Died No. %
Up to 50 years	14	14 100	0 —	0 —	0 —
51–60 years	12	9 75	1 8.3	1 8.3	1 8.3
61–70 years	30	28 93	0 —	0 —	2 7
71-80 years	10	5 50	1 10	1 10	3 30

Results

Of the 66 cases, 56 (84 per cent) made a full recovery. Four patients were left with some disability, which was slight in two and severe in the other two. Six patients died, giving an operative mortaliy of 9 per cent. Thus, of the 60 survivors, 58 made good recoveries. An analysis of the results against the initial level of consciousness shows that patients in semicoma and coma carried a worse prognosis (Table III). A similar analysis against age of the patients showed that patients in the older age group fared worse (Table IV).

DISCUSSION

Some of the difficulties as well as pointers to the diagnosis of chronic subdural haematoma have been revealed in this study. The main difficulty in diagnosis or even suspicion of the lesion is the absence of any history of head injury, or failure to assess the significance of a trivial head injury. It should be remembered that headache and mental symptoms, with relatively few signs in the limbs, are prominent in patients with chronic subdural haematoma. These features are commonly attributed to cerebral infarction or arterio-sclerosis, especially when the traumatic incident has been forgotten. One must be vigilant while making these diagnoses, especially in elderly patients, as in a case of "stroke" further investigations are not usually pursued. In fact, an untreatable lesion ("stroke") should seldom be diagnosed on clinical grounds alone. In a review of the pathological diagnosis of 276 cases diagnosed as "stroke", Ranskind and Weiss (1969) found 16 cases of chronic subdural haematoma.

The source of most chronic subdural haematomata is the bleeding from the communicating veins and the pacchionian granulations near the superior sagittal sinus. The haemorrhage tends to gravitate downwards and the clot is contained by the formation of a membranous sac (Gardner, 1932). Further expansion of the haematoma has been thought to be due to osmotic processes. However, some doubt has been cast on this hypothesis of the pathogenesis of chronic subdural haematoma by the experimental work of Goodell and Mealey (1963).

Skull X-rays may or may not reveal a fracture line. If the pineal gland is calcified it may be displaced from the midline, but with bilateral haematomata it may be central. Occasionally a chronic subdural haematoma may become calcified (Jackson and Clare, 1965). Lumbar puncture not only does not provide any help in the diagnosis, but may be hazardous, and should not be done if a subdural haematoma is suspected, but the case should be referred for neurosurgical opinion.

If available, echoencephalography would demonstrate cerebral hemisphere displacements and the haematoma echoes in the majority of, but not all, cases, and should be over-ruled if findings are contrary to clinical expectation. The final proof of the diagnosis comes from angiography or burr-holes. In the elderly or acutely ill patients, suspected of harbouring chronic subdural haematomata, the diagnosis is best confirmed (or excluded) by exploratory burr-holes. This simple procedure simultaneously diagnoses and, in the majority of cases, cures the condition. In view of the reported 10 to 20 per cent incidence of bilateral lesions the burr-holes must always be made on both sides. Our experience with brain scanning was too limited in this series, but further experience since has shown it to be a very valuable diagnostic procedure in chronic subdural haematomata, but again not absolutely reliable, false negative findings occurring occasionally, even in chronic haematomata with thick membranes.

Isolated reports have appeared documenting spontaneous resolution of chronic subdural haematomata without surgery (Bender, 1960; Gannon, 1962; Gannon et al, 1962), and even of medical treatment by prolonged dehydration (Suzuki and Takaku, 1970). The preferred treatment of chronic subdural haematoma, however, remains surgical in the vast majority of cases.

The results of the treatment depend upon the age and the level of consciousness of the patient (McKissock et al, 1960; Walker et al, 1968). Five of the six patients who died in this series were above the age of 60 years, and all were in coma or semicoma at the time of surgery. The prognosis would, therefore, seem to depend upon the alterations produced locally in the underlying cerebral tissues by the haematoma and the general changes in intracranial dynamics. Because the haematoma is extracerebral and slow in evolution, the latter effects predominate in the form of alterations in the level of consciousness and headaches. The older patients have a worse prognosis because the ageing cerebrovascular state does not allow of these gross disturbances.

SUMMARY

A review of the clinical features, diagnosis and management of 66 cases of chronic subdural haematoma is presented. Difficulties in the initial suspicion of the diagnosis are described. In general, cases of chronic subdural haematoma have a good prognosis, except in elderly persons or those with severely depressed level of consciousness.

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